

Vascular Complications of Pancreatitis: Imaging and Intervention

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Abstract The objective of this study was to highlight technical challenges and potential pitfalls of diagnostic imaging, intervention, and postintervention follow-up of vascular complications of pancreatitis. Diagnostic and interventional radiology imaging from patients with pancreatitis from 2002 to 2006 was reviewed. We conclude that biphasic CT is the diagnostic modality of choice. Catheter angiography may (still) be required to diagnose small pseudoaneurysms. Endovascular coiling is the treatment of choice for pseudoaneurysms. Close clinical follow-up is required, as patients may rebleed/develop aneurysms elsewhere.

Keywords Pancreatitis · Embolization · CT angiography

Introduction

Pancreatitis may cause a spectrum of venous and arterial vascular complications, ranging from asymptomatic venous thrombosis to catastrophic variceal hemorrhage and from incidentally discovered pseudoaneurysms that remain stable over years of follow-up to acute life-threatening rupture.

Hemorrhage is uncommon. Balthazar et al. reported only 26 cases in 1910 patients with pancreatitis over 10 years (1.3%) [1]. Hemorrhage is usually due to erosion of a major pancreatic or peripancreatic vessel with massive bleeding into the gastrointestinal tract or abdominal cavity

or to formation and subsequent rupture of an arterial pseudoaneurysm.

Diffuse bleeding may also occur with extensive necrosis (Fig. 1) or by thrombosis of portal/splenic/superior mesenteric veins, resulting in compartmental portal hypertension with gastric, mesenteric, or colonic varices. Venous complications are not uncommon. Mortelet et al, retrospectively reviewed 100 consecutive patients with acute pancreatitis. Arterial hemorrhage was seen in 5% of patients, and venous abnormalities, including splenic, superior mesenteric, and portal vein thrombosis, were seen in 19%, 14%, and 13% of patients, respectively [2].

Bergert et al. reported severe bleeding complications in 36 of 541 patients with chronic pancreatitis, a prevalence

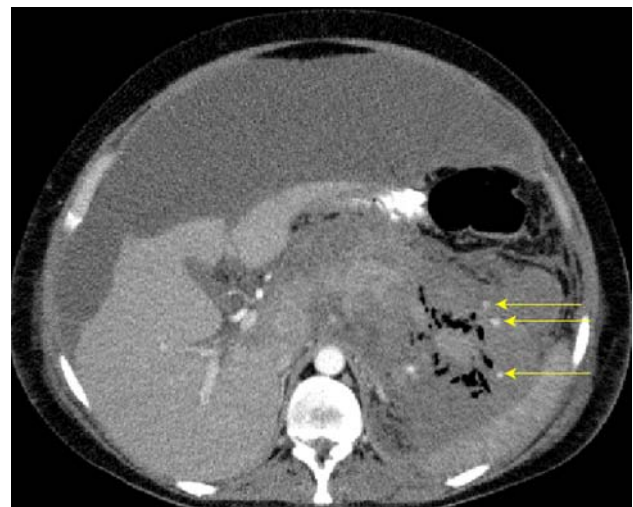


Fig. 1 A 41-year-old woman with acute pancreatitis. CT demonstrates hemorrhagic necrosis with multiple focal areas of hemorrhage (arrows). Despite coil embolization of the splenic artery the patient still required 10 units of packed red cells at necrosectomy

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of 6.7%. The most common cause of major hemorrhage was pseudoaneurysm, in 25 patients (69.4%). Hemorrhage was caused by ulceration or variceal bleeding in 22.2%, and 8.4% of patients demonstrated splenic infarction or rupture [3].

Remote vascular complications may also occur; these include intestinal ischemia/necrosis (Fig. 2), subcutaneous fat necrosis, and vascular retinopathy [4].

Refinements in cross-sectional imaging and endovascular technique have led to an increasingly important role for the diagnostic and interventional radiologist in the management of these patients. This paper reviews imaging findings and highlights several potential pitfalls in imaging and intervention.

Pathophysiology

Several pathobiologic responses occur in acute pancreatitis including edema, inflammation, and various forms of parenchymal cell injury including vasospasm, necrosis, and apoptosis. Hypovolemia, hypotension, and sympathetic stimulation are major causes of vasospasm. Local inflammation, endothelin, and complement system activation can contribute to ischemia/thrombosis. The microcirculation has been demonstrated to play an important role in mediating the severity of and necrotizing response in acute pancreatitis [4, 5]. Animal studies have demonstrated that spinal nerves contribute to the regulation of pancreatic blood flow during pancreatitis.

Leakage of pancreatic enzymes from an inflamed pancreas may result in enzymatic autodigestion of vascular walls with diffuse bleeding, in association with extensive necrosis, or pseudoaneurysm formation. The wall of a pseudocyst can also incorporate a visceral artery, converting it into a pseudoaneurysm (Fig. 3). The splenic artery is the most common artery involved (60%–65%), due to its contiguity with the pancreas, followed, in

decreasing order of frequency, by gastroduodenal (20%–25%), pancreaticoduodenal (10%–15%), hepatic (5%–10%), and left gastric arteries (2%–5%) [7]. Bleeding may occur into the peritoneal (Fig. 4) or retroperitoneal space or the pancreatic duct (hemorrhage pancreatitis)—a well-recognized but rare cause of occult GI blood loss.

Severe hemorrhage may also be associated with the development of pancreatic and peripancreatic abscesses. This process may take place over weeks or months following a severe acute episode.

A further pathogenic mechanism relates to thrombosis of the splenic vein secondary to severe contiguous inflammation, necrosis, or abscess, or by pseudocyst compression. As the splenic artery remains patent the pressure rises in the collateral (short gastric and pancreaticoduodenal) outlet veins. Sinistral portal hypertension with esophageal and gastric varices may develop and cause life-threatening hemorrhage [6].

Imaging

The use of noninvasive imaging modalities, including ultrasound (US), duplex Doppler US, and bolus-dynamic CT, and earlier use of diagnostic and therapeutic angiography have led to significant reductions in morbidity and mortality rates for patients with vascular complications from pancreatitis [8].

Ultrasound

Characteristic appearance of turbulent arterial flow within an anechoic mass is indicative of pseudoaneurysm. Color duplex US should be used in the evaluation of all peripancreatic fluid collections during the initial US examination to avoid misinterpretation of an aneurysm or a pseudoaneurysm as a simple or complex fluid collection.

Fig. 2 A 41-year-old woman with acute pancreatitis, duodenal and colonic necrosis with colcutaneous and gastrocutaneous fistulae. Contrast studies demonstrate duodenal (A) and sigmoid colon perforation (B). Note inferior vena cava filter and coils from prior embolizations

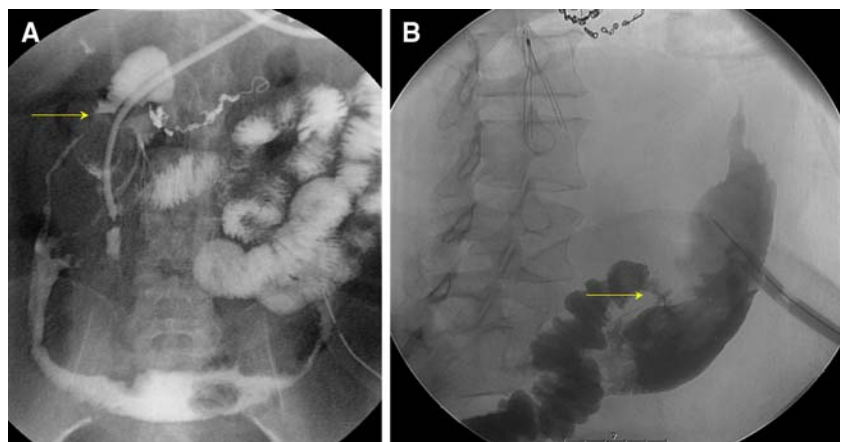
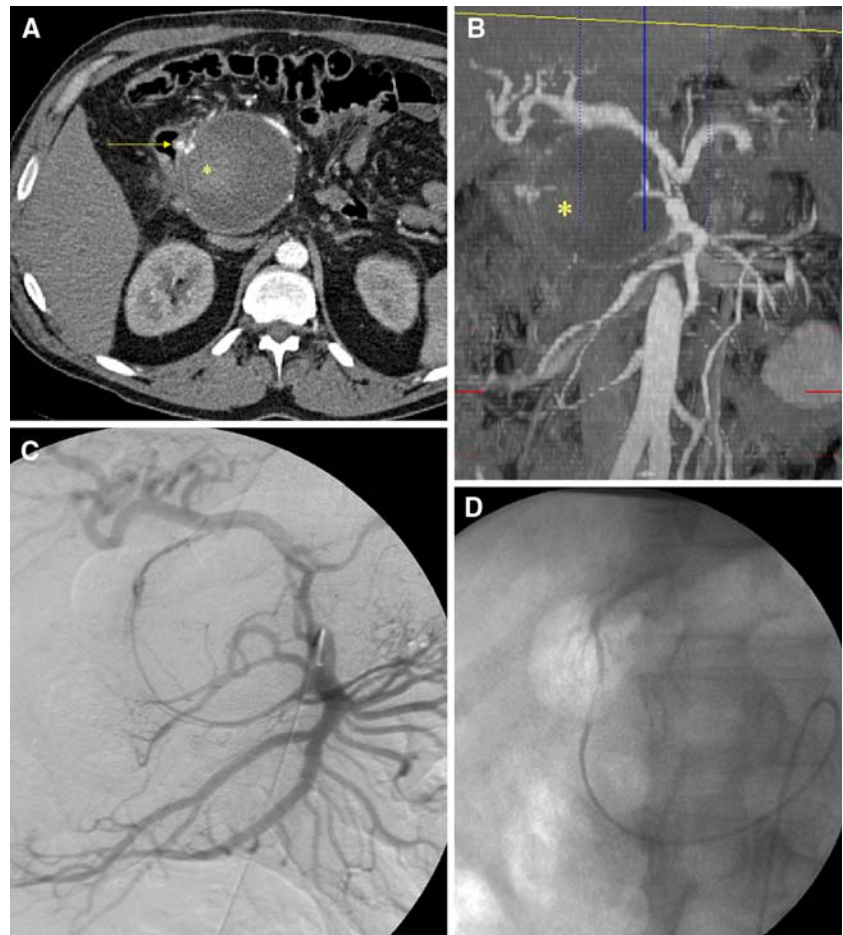


Fig. 3 A 44-year-old man with acute pancreatitis and melena, hemodynamically unstable. Axial CT (A) with coronal MIP reconstruction (B) demonstrated a mixed attenuation pseudocyst containing eccentric high-density material, consistent with blood (*). The GDA was stretched around the pseudocyst and a small pseudoaneurysm was present between the lateral cyst wall and the duodenum (arrow). Thus directed, the SMA was selected (C). There was active bleeding from the inferior pancreatic/duodenal arcade, with contrast extravasating into the duodenal lumen (D). The IPDA was coiled from below. A microcatheter was required to access the hepatic artery to allow coiling of the GDA from above (not shown)



Doppler US may also demonstrate portal venous thrombosis with development of varices. Although calcification and local ileus may limit its sensitivity, contrast-enhanced US (CEUS) has been recommended as a first-choice imaging procedure for the assessment of patients with acute pancreatitis, especially when iodinated contrast medium injection is contraindicated [9]. Rickes et al. found a very good correlation ($r = 0.807$, $p < 0.01$) between the CT severity index (CTSI) and a US severity index, developed by the authors, in 31 patients with acute pancreatitis. Using CT as the gold standard, the sensitivity, specificity, and positive and negative predictive values of CEUS for detecting severe acute pancreatitis (CTSI ≥ 3) was 82%, 89%, 95%, and 67%, respectively. The limitations of CEUS lay in detection of peripancreatic fatty tissue necrosis [9].

Computed Tomography

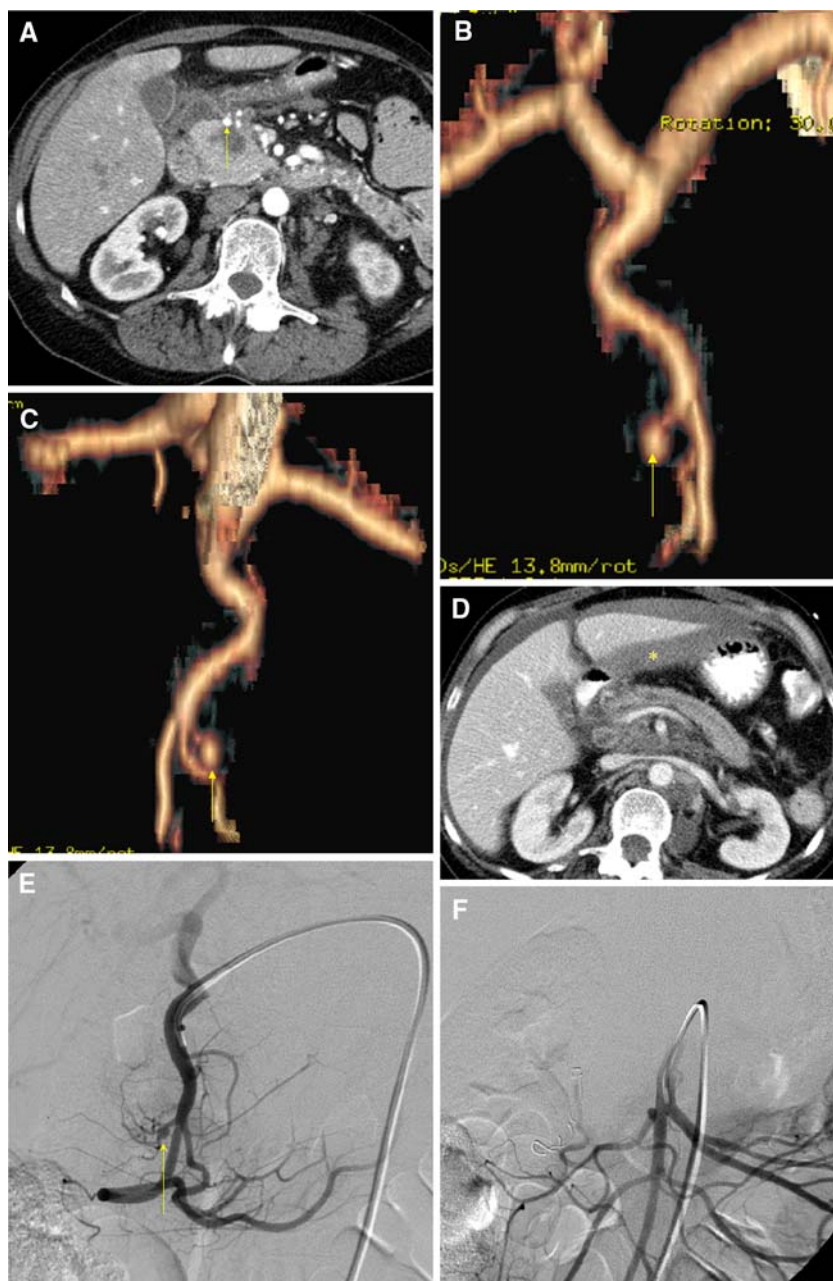
Multislice helical CT (MSCT), with its multidetector technology and faster rotation times, has led to new dimensions in spatial and temporal resolution in CT

imaging. In contrast to single-slice CT, thinner-slice collimations covering longer anatomic volumes allow for increased spatial resolution along the z -axis and almost-isotropic voxels with high-quality multiplanar and three-dimensional image reconstructions (Fig. 5). The high speed of multislice CT allows organ imaging in clearly defined perfusion phases, e.g., arterial, parenchymal, and portal venous phases. Contrast agents with higher iodine concentrations (400 compared with 300 mg iodine per milliliter) led to improved vascular enhancement.

In the early 1990s, Balthazar et al. developed criteria for assessing the severity of pancreatitis, based on the presence or absence of pancreatic and peripancreatic edema/inflammation, the degree of necrosis, and extrapancreatic fluid collections [10].

Mortele et al. retrospectively reviewed 159 contrast-enhanced helical CT scans of 100 consecutive patients with acute pancreatitis. Severity was graded as mild ($n = 59$), moderate ($n = 82$), or severe ($n = 18$). There was no statistically significant correlation between the prevalence of “vascular abnormality” and the severity of pancreatitis using the CTSI, even on subgroup analysis, however the complications were predominantly venous thromboses [2].

Fig. 4 A 47-year-old female with a history of scleroderma and chronic pancreatitis. A small GDA aneurysm was identified on CTA (arrows, Fig. 4A–C). The patient later presented with an “acute abdomen”, and CT demonstrated hemoperitoneum (Fig. 4D (*)). The GDA was coiled from above, across the pseudoaneurysm. Check SMA angiogram confirmed no perfusion from IPDA (Fig. 4E,F). [Case courtesy of Dr M Voss, St Joseph’s Hospital, Hamilton, Ontario]



In contrast, the CTSI was recently shown to have greater sensitivity, specificity, and positive and negative predictive values than the Acute Physiology and Chronic Health Evaluation (APACHE-II) score, C-reactive protein ≥ 150 mg/L, and Ranson criteria in predicting acute pancreatitis outcome [11, 12].

Multislice CT angiography (CTA) is a sensitive and accurate technique for the detection of major arterial hemorrhage in inflammatory pancreatic disease and should be considered as the first investigation in diagnosis and for planning intervention. Image acquisition is in the arterial phase when pancreatic parenchymal and portal venous

enhancement is relatively weak. Optimal imaging is facilitated by automated bolus tracking, using a low threshold for aortic attenuation of 80 HU and the shortest possible scan delay. High-quality maximum intensity projection reconstructions can direct the interventionalist to an acutely bleeding vessel, saving time and contrast (Fig. 6).

Unstable patients with massive GI bleed may proceed directly to angiography. For most of these patients the bleeding vessel will be readily apparent angiographically. We have encountered one patient with severe atherosclerosis in whom the origins of the celiac trunk and superior mesenteric artery (SMA) were particularly atherosclerotic.

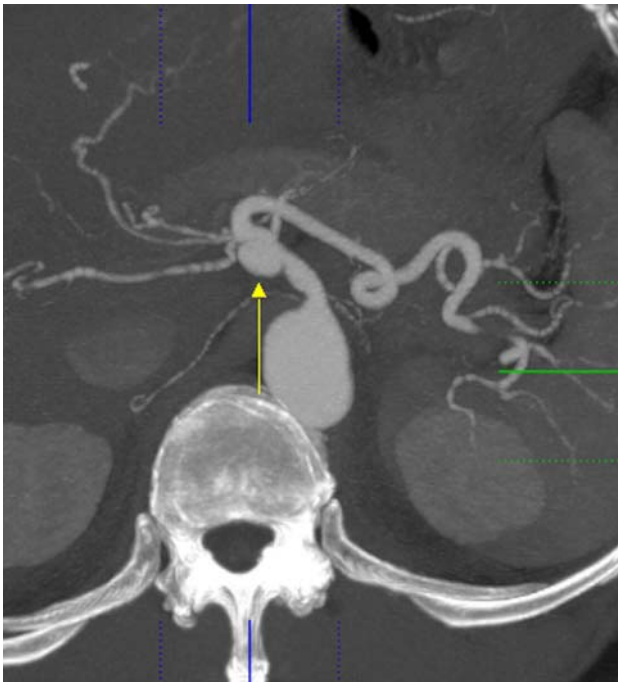


Fig. 5 A 54-year-old male. Small stable aneurysm of posterior celiac artery (arrow). This was followed with CTA with no requirement for angiography

CT arteriography localized the bleeding to a splenic artery–transverse colon fistula and facilitated microcatheter access across the celiac trunk and successful coil embolization (Fig. 7).

Angiography

Digital subtraction angiography (DSA) has long been the gold standard for the detection of a visceral artery pseudoaneurysm or for the site of active bleeding in patients with pancreatitis. Dilatation of the intrapancreatic branch arteries and homogeneous parenchymal staining of the pancreatic gland have been described as characteristic for acute edematous pancreatitis, whereas mottled, heterogeneous staining and ischemic change with narrowing, sudden tapering, and a beaded appearance of the intrapancreatic branch arteries, consistent with vasospasm, are most commonly observed in acute necrotizing pancreatitis [5].

Computed Tomographic Angiography or Digital Subtraction Angiography?

With the evolution of CTA it is likely that catheter angiography will increasingly become a therapeutic tool rather than a purely diagnostic one. Hyare et al. retrospectively

compared 29 studies in 25 patients with major bleeding as a complication of pancreatitis in which CTA was performed within 24 h preceding DSA. The sensitivity and specificity of CTA for the detection of major arterial bleeding on a background of pancreatitis were calculated as 0.947 and 0.900, respectively [13]. Very small pseudoaneurysms may occasionally be missed on CTA, and angiography remains the gold standard (Fig. 8).

Nicholson et al. reported that 5 of 23 pancreatitis-related aneurysms (22%) either were undetectable or appeared only in the late venous phase of angiography—these were thought to arise from the vascular bed, rather than a major artery [14]. It was not clear how well this subgroup was visualized on CTA. We recommend a biphasic protocol with a portal venous phase, as small pseudoaneurysms and those with narrow necks may become significantly more conspicuous during portal venous phase imaging (Fig. 9). This additional phase is also useful in the evaluation of venous thrombosis that can be missed if CTA alone is performed (Figs. 10–12).

Takeda et al. compared angiography with contrast-enhanced CT (CE-CT) in 102 patients with acute necrotizing pancreatitis (ANP) [5]. A poorly perfused/low-density area in the pancreas on admission CE-CT was considered to represent ANP. Ischemic change with vasospasm of intra- and extrapancreatic arteries corresponded with areas of poor perfusion detected by CE-CT. The extent of ischemic change correlated with the extent of poorly perfused pancreas and mortality [5]. Progression from vasospasm to occlusion of intrapancreatic branches, consistent with evolution from pancreatic ischemia into necrosis, was seen in some cases, whereas improvement of ischemic change/vasospasm was confirmed by follow-up angiography in those who recovered uneventfully [5].

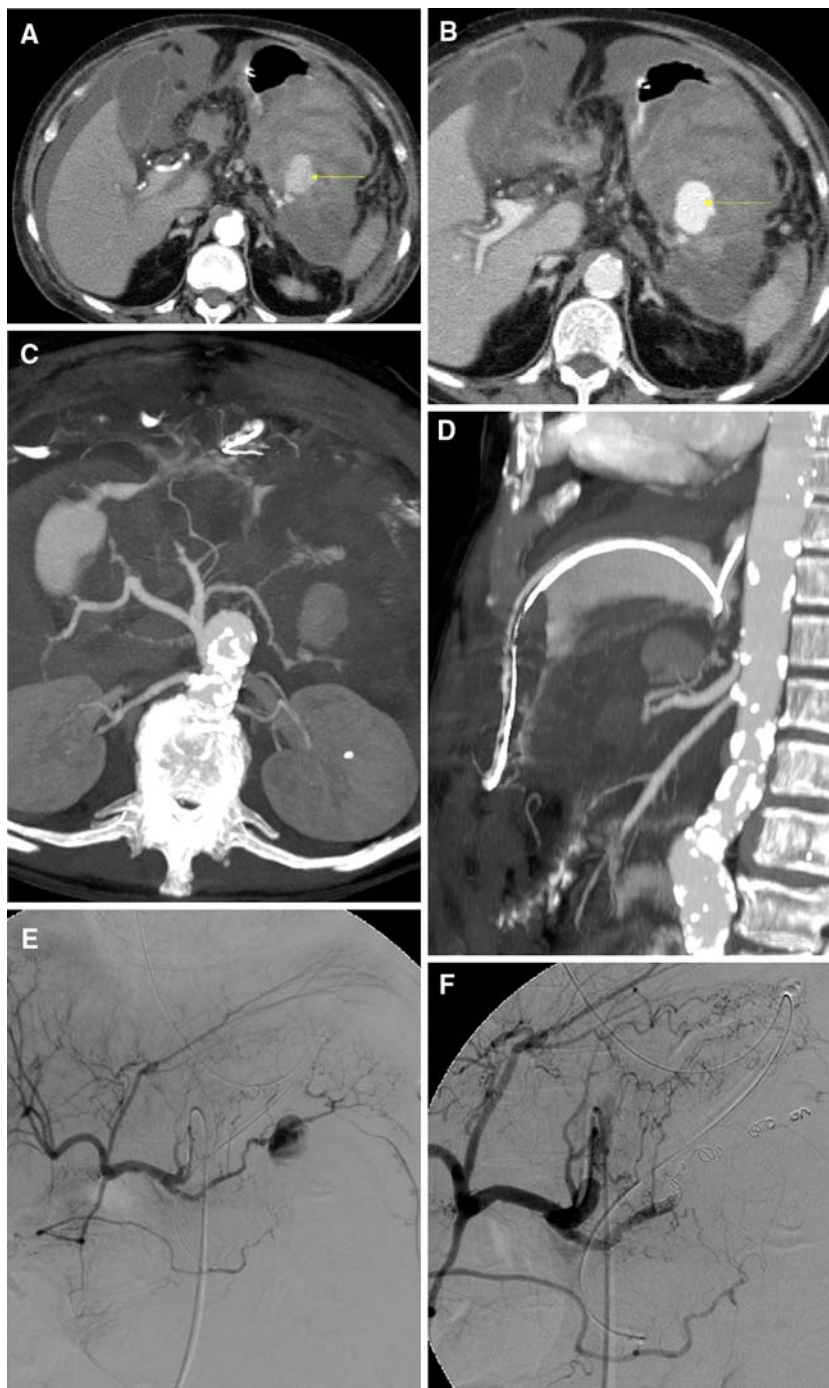
Transcatheter Arterial Embolization

The traditional surgical treatment for pseudoaneurysms included celiotomy, ligation of the celiac trunk, and partial pancreatectomy. Transcatheter arterial embolization provides a minimally invasive, effective alternative [14–17]. Embolizing the splenic artery prior to surgical necrosectomy may significantly improve intraoperative blood loss for patients with hemorrhagic necrosis. It may also be useful prior to surgery to reduce left upper quadrant varices if there is splenic venous thrombosis [6].

Coagulopathy, sepsis, and renal failure are relative contraindications to percutaneous transcatheter embolization. Appropriate efforts should be made to correct or improve these conditions prior to the procedure.

Typically, embolization can be performed through a short 5-Fr common femoral sheath, however, a long sheath

Fig. 6 A 79-year-old woman with known pancreatic pseudocyst and recent duodenal ulcer. Acute deterioration in ICU. CT demonstrated ruptured pseudoaneurysm of the splenic artery (arrows, Fig. 6A-D). Multiplanar reconstruction allowed for precise anatomic location. Following embolization (Fig. 6E,F), she required laparotomy for abdominal compartment syndrome (bladder pressures >40mmHg) and subsequently underwent pancreatic necrosectomy. She died 3 weeks later from multiorgan failure



should be considered in patients with atheromatous disease involving the abdominal aorta or iliac arteries, reducing potential vessel wall injury during catheter exchange, and, in an ectatic aorta, provides stability for coaxial access.

CTA should obviate the need for abdominal aortography to delineate the arterial anatomy. Subselective angiography, longer injection durations with imaging into the venous phase, and use of carbon dioxide for contrast medium can improve sensitivity for small bleeds. CO₂ has a low viscosity, lacks a significant capillary phase to

obscure small hemorrhages, and does not mix with extravasated blood, avoiding dilution.

Successful embolization requires getting as close to the bleeding vessel as possible. The celiac axis and SMA can be selected with a variety of catheters. A 4-Fr hydrophilic Cobra catheter (e.g., Glide Catheter; Terumo/MediTech, Boston Scientific, Waltham, MA) may be sufficient for access and embolization, however, a microcatheter is often required for access into small tortuous vessels. “Parking” a 5-Fr Cobra 2, Simmons 2, Sos-Omni, or other

Fig. 7 A 76-year-old man with chronic pancreatitis and acute, bright-red rectal bleeding. He had severe atheroma and we were unable to access the splenic artery on initial angiography. CT arteriportography before (A) and after (B) IV contrast demonstrated a large splenic artery pseudoaneurysm (*) eroding into the distal transverse colon. The splenic artery was eventually catheterized using a microcatheter and successfully coiled

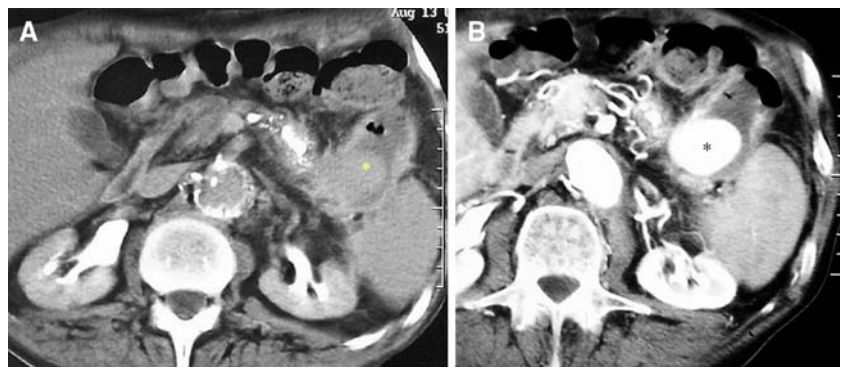


Fig. 8 A 9-year-old boy with chronic pancreatitis and recurrent pseudocyst. Episodic abdominal pain from age 9 months; multiple investigations, including ERCP, laparoscopy, CT, and MRI. Recent significant episodic GI blood loss (20 units of blood over 6 months). Ultrasound (A, B) demonstrated a small pseudocyst (arrow) lateral to the SMA. CTA (C, D) was followed by catheter angiography (E): two tiny pseudoaneurysms from the SMA at the level of the pseudocyst (arrows) were identified. These were oversewn at Whipple procedure (F). The resected specimen demonstrated pseudocyst and chronic pancreatitis. There was no pseudocyst recurrence, further bleeding, or pain at 6-month follow-up

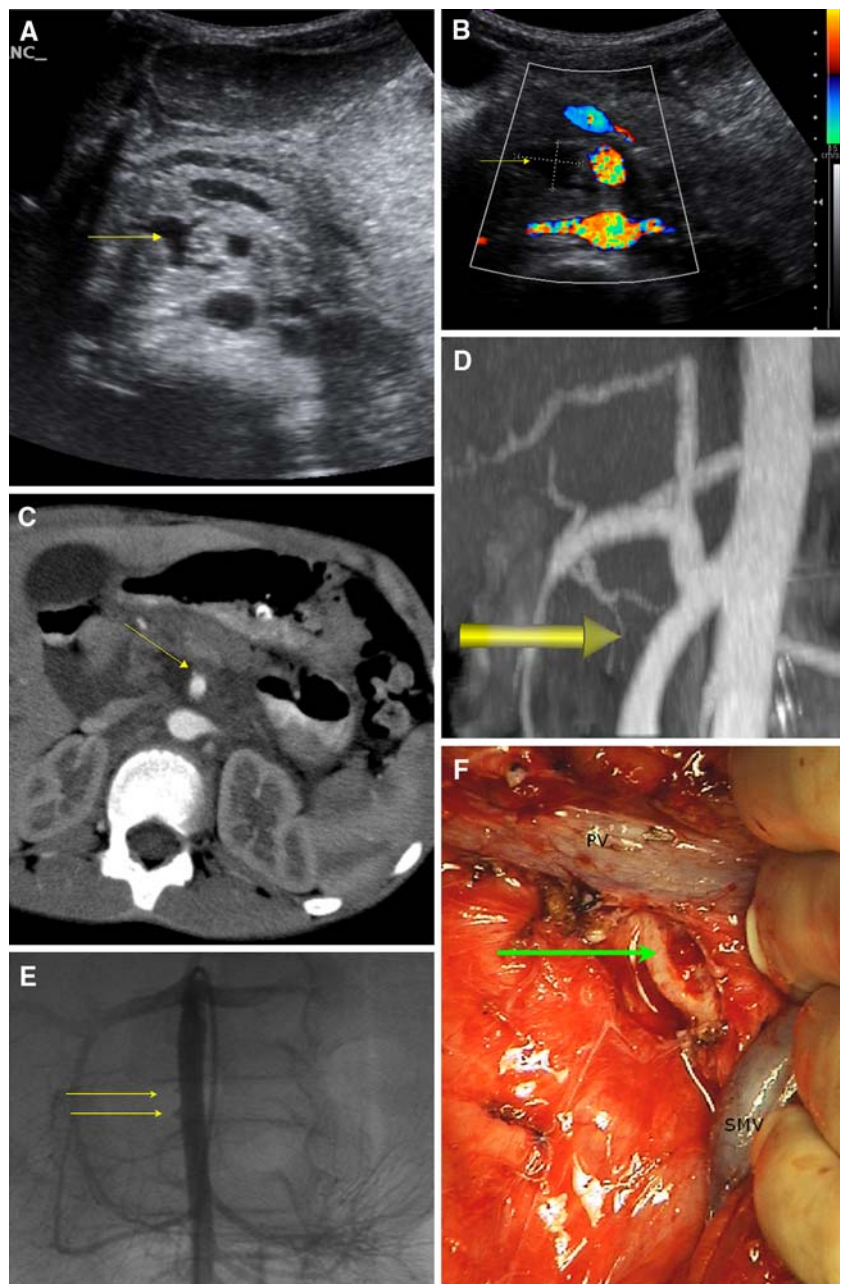


Fig. 9 An 83-year-old woman with complicated gallstone pancreatitis. Arterial and portal venous phase CT (A, B) demonstrated a slow-filling hepatic artery pseudoaneurysm, most clearly seen in the portal phase (arrows), and a moderate pancreatic pseudocyst. CT significantly aided the radiologist in this case, as the aneurysm was difficult to visualize on angiography. The right hepatic artery was coiled

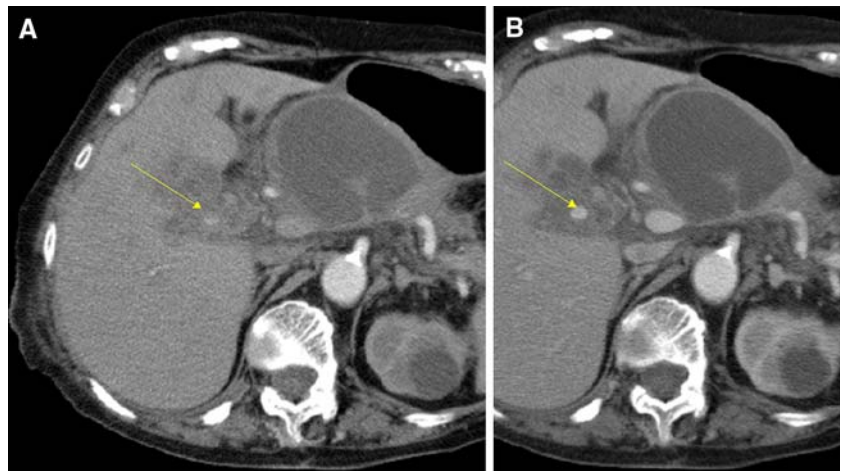


Fig. 10 A 45-year-old man with acute pancreatitis. Segmental SMV thrombus (arrows; A, B). Note how much more conspicuous this is in the portal venous phase (B)

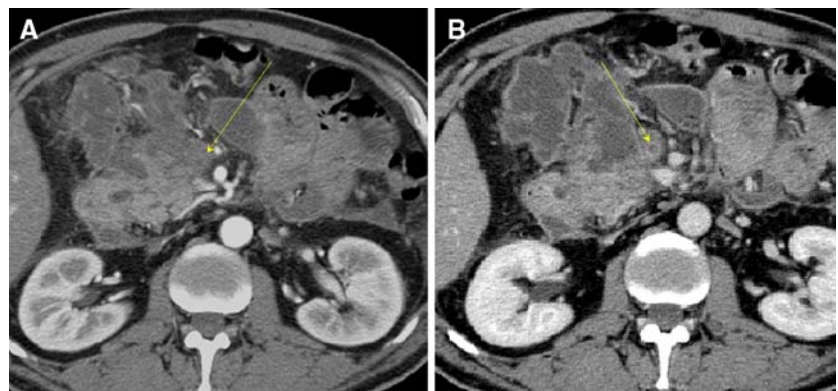


Fig. 11 A 47-year-old female with acute pancreatitis complicated by GDA aneurysm. History of scleroderma. Portal venous phase CT demonstrates resolving pseudocysts and left renal vein thrombosis (*). This may have been overlooked if a CTA protocol alone was used.

appropriately preshaped catheter in the proximal arterial trunk can provide sufficient stability for microcatheter access. Engaging the left gastric artery may be a technical

challenge. A Waltman loop or reverse curve catheter may be required. Occasionally an angled catheter and hydrophilic guidewire may be sufficient.

There is an array of microcatheters available for selective and superselective access. These are typically 3-Fr-outer diameter, with 0.021- to 0.027-in. inner diameter. Catheters with a larger inner lumen allow for higher flow rates, making them more suitable for infusion and perhaps less likely to occlude with glue. Most microcatheters have one or two radiopaque markers at the distal tip. There is also a variety of guidewires, several of which are steerable with a torque device and hydrophilic.

Occasionally access may be impossible due to acute angulation of the feeding vessel. In this situation consideration can be made of using a reverse curve catheter or switching to an axillary approach (Fig. 13).

Basic tenets of aneurysm embolization apply. Proximal and distal occlusion of the artery bearing the pseudoaneurysm is desirable. In the case of the splenic artery, placing the distal coil as close to the aneurysm as possible improves the chances of reconstitution of arterial inflow to the spleen, thereby reducing the risk of splenic infarction.

Digital subtraction roadmapping may reduce the need for repeat injection of contrast medium to demonstrate the

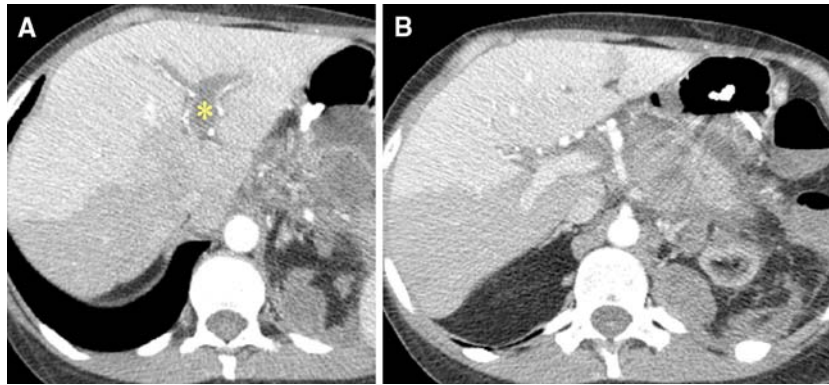
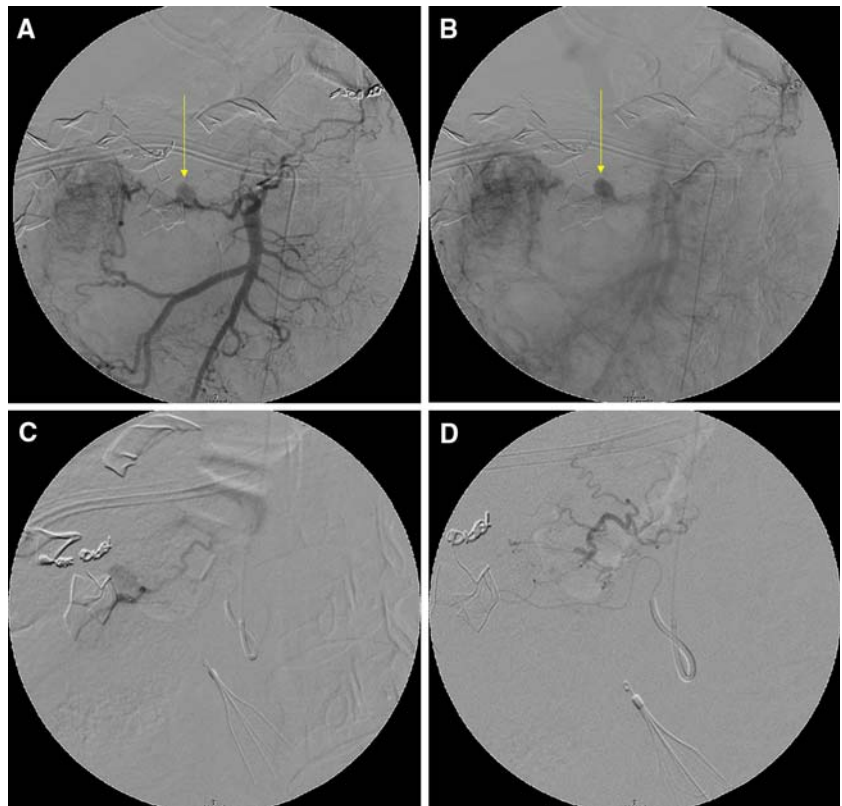


Fig. 12 A 33-year-old female with acute pancreatitis and left portal vein thrombosis (*). Hyperattenuation in the region of thrombosis results from compensatory increased hepatic arterial flow and lack of contrast material dilution during the arterial phase because of

diminished, nonopacified early portal venous return. Complete portal vein thrombosis can result in diffuse peripheral hyperenhancement if central cavernous transformation has developed

Fig. 13 A 42-year-old female with acute pancreatitis. SMA injection demonstrated an acute pseudoaneurysm of the middle colic artery (arrows; **A, B**). We were unable to access the feeding artery from the right femoral route. Following left axillary access, a 5-Fr Sos-Omni catheter was formed in a jejunal branch, allowing microcatheter access across the pseudoaneurysm (**C, D**).



position of the wire/catheter in relation to the vessel lumen. Buscopan or glucagon should be considered if there is excessive bowel peristalsis.

Embollic Agent

Fibered coils are made up of helically wound steel, platinum, or tungsten wires, with attached synthetic fibers to maximize thrombogenicity, and are the mainstay embolic

material used for pseudoaneurysm occlusion. They are available as straight or as complex tertiary preformed shapes such as helical and tapered helical, with a range of sizes from 1 to 20 mm to match the vessel diameter. Coils are generally manufactured in either a 0.018- or a 0.035-in. system to facilitate deployment through a microcatheter or a 5-Fr catheter. It should be noted that the 0.018-in. coils are optimally deployed through a microcatheter with a 0.021-in. inner diameter, as the coils can become wedged between the pusher wire and the catheter wall when used in

a 0.027-in. or larger “high-flow” catheter. The coil may still be deployable by saline flush, which can be expeditious and may achieve a more distal embolization from the microcatheter tip; however, the use of a pushing wire may be more precise and thus preferable for deploying the initial coil.

Smaller diameter coils (0.014 and 0.010 in.) designed for intracranial applications are also available and may offer the option of detachability and retrievability. Detachable coils are affixed to the delivery wire and can be detached by a threaded, pneumatic, spring-loaded, or electrolytic mechanism of deployment. If a detachable coil is positioned suboptimally, it can be easily retracted and repositioned. These coils generally do not have thrombogenic fibers to facilitate occlusion of the vessel.

It is not necessary to fill the aneurysm sac, as long as the efferent and afferent arteries are occluded and the potential for reperfusion is minimized. Packing the sac with coils increases the risk of rupture, is an unnecessary expense, and is time-consuming. In addition, the insertion of the coils into the sac makes follow-up imaging to assess reperfusion very difficult.

Liquid embolic materials such as glue can provide effective embolization and might be considered when a pseudoaneurysm can be entered but not crossed. *N*-Butyl cyanoacrylate glue (NBCA) polymerizes immediately upon contact with an ionic medium such as saline or blood. A strict nonionic environment is therefore required for preparation. NBCA causes complete vessel occlusion, giving improved hemostasis during coagulopathy. It is nonradiopaque and is typically mixed with ethiodized oil (Lipiodol). An assessment of flow rate across the pseudoaneurysm is required to allow calculation of glue:contrast ratio, as this affects viscosity and polymerization rates. While this makes it technically more difficult to control than coils, it facilitates occlusion distal to the microcatheter tip. Care must be taken when injecting through a microcatheter, as there is potential for catheter occlusion or entrapment and loss of access. Visceral pseudoaneurysms have been embolized successfully using both NBCA and, more recently, ethylene-vinyl alcohol.

Gelfoam has been used by some authors [8], however, it may be rapidly resorbed by pancreatic proteolytic enzymes and has the potential to migrate into small arteries, potentiating tissue ischemia.

Direct percutaneous injection of thrombin has been described in several case reports and should be considered where there is technical difficulty with the endovascular route or when the aneurysm is not visualized despite selective angiography. More than one injection may be required [14].

Occlusion balloons cause proximal arterial occlusion with potential for collateral flow and continuing

pseudoaneurysm perfusion. Limited use as a temporizing measure (presurgical) has been described [8].

Placement of covered stents across visceral artery pseudoaneurysms, with preservation of distal flow, has become technically feasible in recent years with smaller and more flexible delivery systems. The use of stent-grafts remains limited by anatomic suitability and risks of stent migration, thrombotic occlusion/intestinal ischemia, and potential infection. Hyare et al. placed four stents in patients with arterial complications of pancreatitis, three in the common hepatic artery. Misplaced stents involving the left phrenic artery and celiac axis occurred in two patients [25].

Results with Embolization

Rebleeding is a potential problem with endovascular management. In a multicenter retrospective study of 104 cases with arterial lesions associated with pancreatitis, Boudghene et al. reported a success rate of 78%, with rebleeding in 37% from 32 patients treated by embolization [7]. Data from recent case series are more encouraging, although published studies are limited to small retrospective case series (Table 1). Bergert et al. reported a higher rebleeding rate after surgery (25%; $N = 16$) versus embolization (11%; $N = 9$) for pancreatitis-related pseudoaneurysms. Two patients died after surgery, and one after primary embolization (8.3% mortality) [3].

Golzarian et al. evaluated transcatheter embolization as a therapeutic modality in nine patients with 13 pseudoaneurysms secondary to pancreatitis. Eight patients had chronic pancreatitis. In nine patients the pseudoaneurysms were successfully isolated from the circulation, and hemostasis was achieved. Rebleeding occurred in two patients, 18 and 28 days after embolization, respectively, and was successfully treated by repeated embolization. One patient with severe pancreatitis died from sepsis 28 days after embolization [18].

Nicholson et al. reviewed 23 patients with aneurysms or pseudoaneurysms associated with acute pancreatitis, treated by endovascular or percutaneous methods. All underwent CT and angiography. Nineteen patients were treated by primary coil embolization, two early recurrences. Four patients were treated by primary percutaneous thrombin injection; all four exhibited late recurrences but were successfully treated by further percutaneous thrombin injections. Twenty-one patients (91.3%) were alive at 6 months [14].

Deshmukh et al. evaluated transcatheter embolization as a therapeutic modality in 30 patients with visceral pseudoaneurysms secondary to pancreatitis. In 29 patients the pseudoaneurysms were successfully isolated from the circulation, and hemostasis was achieved. One patient

Table 1 Rebleeding rates and mortality following endovascular intervention of hemorrhagic complications of pancreatitis

Year	Author	No. of patients	Rebleed	Endovasc Rx definitive	Mortality	Follow-up
1993	Boudghene [7]	32	37%	78%	16%	
1993	Savastano [20]	5	80%	60%	20% (rebleeding)	
1997	Golzarian [18]	9	22%	100%	11% (sepsis)	
1997	Gambiez [21]	14	21%	79%	7%	
1999	DePerrot [22]	3	33%	66%	33% (rebleeding)	
2000	Carr [23] ^a	6	16%	50%	0%	44 mo
2003	Beattie [24]	11	27%	73%	27%	
2004	Deshmukh [15]	30	6.1%	100%	3% (sepsis)	
2004	Bergert [3]	9	11% (25% for surgery)	89%	8.3%	Median 4.6 yr
2005	Mendelson [16]	9	22%	100%	11% (sepsis)	
2006	Nicholson [14]	23	10% coils, 100% thrombin	100%	0%	91% alive at 6 mo
2007	Hyare [25]	27		80%	20%	

^a An additional 4 patients underwent endovascular intervention following failed first line surgery, 3 of whom died

underwent re-embolization for rebleeding. Twenty-nine patients improved clinically. One patient in whom the pseudoaneurysm was successfully embolized died due to septicemic shock [15].

Mendelson et al. reported embolization in 13 pseudoaneurysms in nine patients. Two patients who rebled had successful reembolization. One patient died from sepsis, 28 days postembolization [16].

Hyare et al. recently reported a series of 35 patients with major arterial complications of pancreatitis, 27 of whom underwent angiographic intervention. Twenty-eight percent of the patients had acute necrosis. A technical failure rate of 19.5% included three patients in whom the bleeding site could not be accessed. Overall mortality was 20%. Empiric arterial embolization was performed in six patients, with clinical success in four, and the authors have highlighted the need for a prospective study to further evaluate prophylactic embolization in patients with negative angiography [25].

Complications from embolization are uncommon. Splenic infarction is not rare but is typically clinically silent and usually managed conservatively. Boudghene et al. and Mauro et al. have reported splenic and intestinal necrosis [7, 17]. Vascular dissection, coil migration, and misplaced stents have also been reported [25]. Aneurysm rupture during embolization is rare. We have observed pancreatic duct opacification in one patient following iatrogenic microcatheter dissection of a splenic artery aneurysm (Fig. 14).

Bleeding into the pancreatic duct (hemosuccus pancreaticus) is rare. Dasgupta et al. reported five patients over 8 years. Direct communication with the pancreatic duct was seen in three of five at angiography and all had successful coil embolization as primary therapy, with additional PVA in two patients and glue in one patient [19].

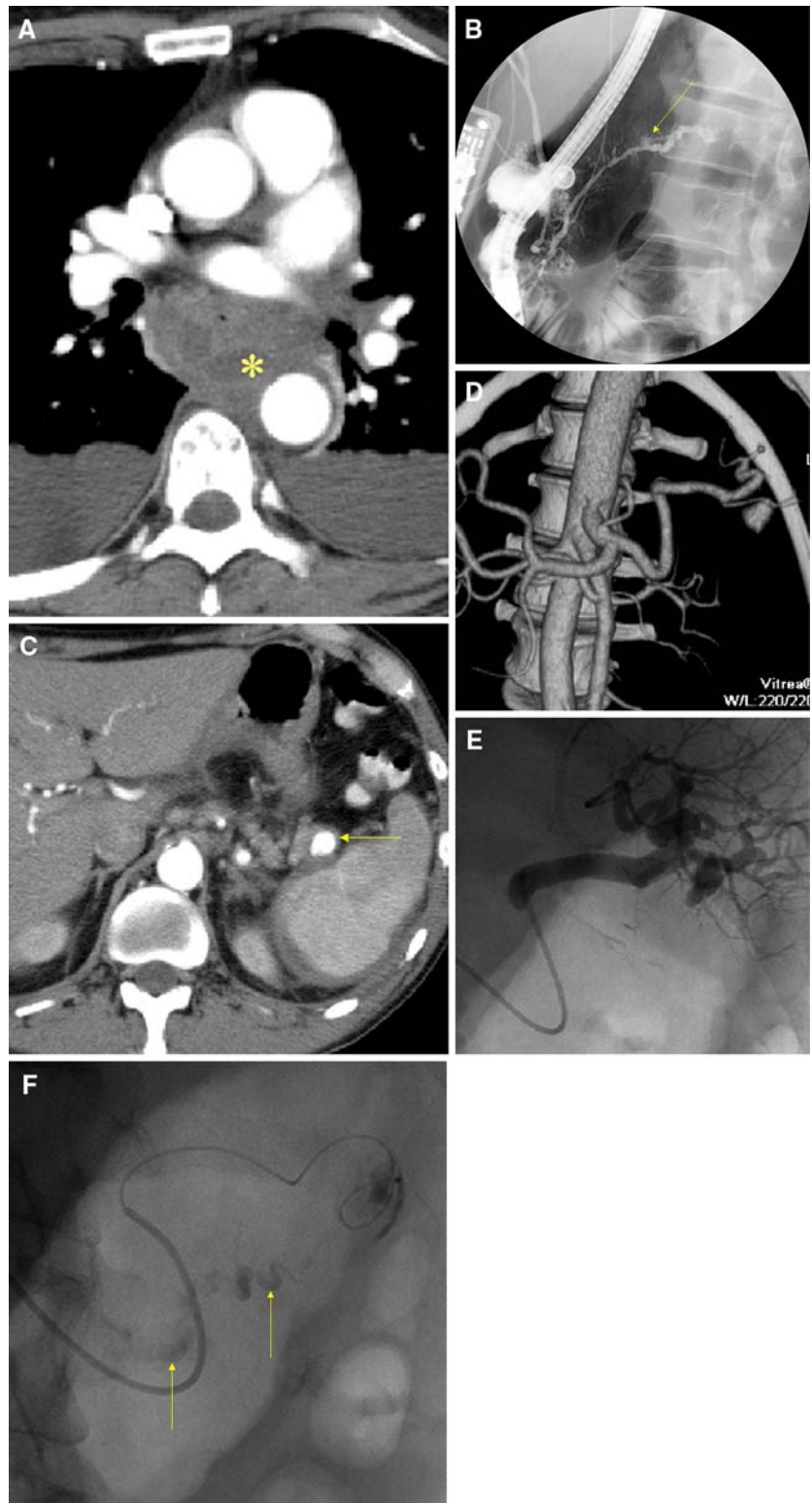
Follow-up imaging must be considered, particularly if a patient fails to clinically improve or has further bleeding. CT must be carefully evaluated, changing viewing windows as necessary, as artifact from coils may obscure pathology (Fig. 15). A precontrast study is required as dense old blood/thrombus within the aneurysm can be mistaken for enhancement.

Aneurysms may also develop at different sites in the same patient. In a single patient, we have embolized the splenic artery prior to necrosectomy and a concomitant right gastric artery pseudoaneurysm, followed 2 weeks later by a middle colic artery pseudoaneurysm and 4 weeks later by a small unnamed pancreatic artery pseudoaneurysm, the latter accessed through a small collateral from the SMA, using a microcatheter. The patient later bled from retroperitoneal venous hypertension.

Take-Home Points

- Be alert for arterial and venous complications, especially small pseudoaneurysms.
- Use color Doppler US; consider contrast-enhanced US if the patient is unable to have CTA.
- Both arterial and delayed phase CT are required, particularly for slow-flow aneurysms.
- Consider CTA/portography if the anatomy/source is unknown.
- Catheter angiography may (still) be required to diagnose small pseudoaneurysms.
- Endovascular coiling is the treatment of choice.
- Remember the basic principles of front- and backdoor embolization; consider glue.

Fig. 14 A 46-year-old male with acute-on-chronic pancreatitis complicated by acute mediastinitis secondary to pancreaticomediastinal fistula (*; **A**). ERCP demonstrates fistula origin from the pancreatic main duct (arrow; **B**). Splenic artery aneurysm noted on CTA (**C, D**) and DSA (**E**). Iatrogenic dissection with filling of the pancreatic duct during microcatheter manipulation (arrows; **F**). The patient was asymptomatic and the aneurysm was eventually coiled.



- Remember axillary/brachial access.
- Use a microcatheter system sooner rather than later.
- Patients must be followed up closely, as they may rebleed or develop aneurysms elsewhere.

- Follow-up imaging requires both unenhanced and enhanced studies.

In conclusion, pancreatitis may cause a spectrum of venous and arterial vascular complications. US, CT, and catheter

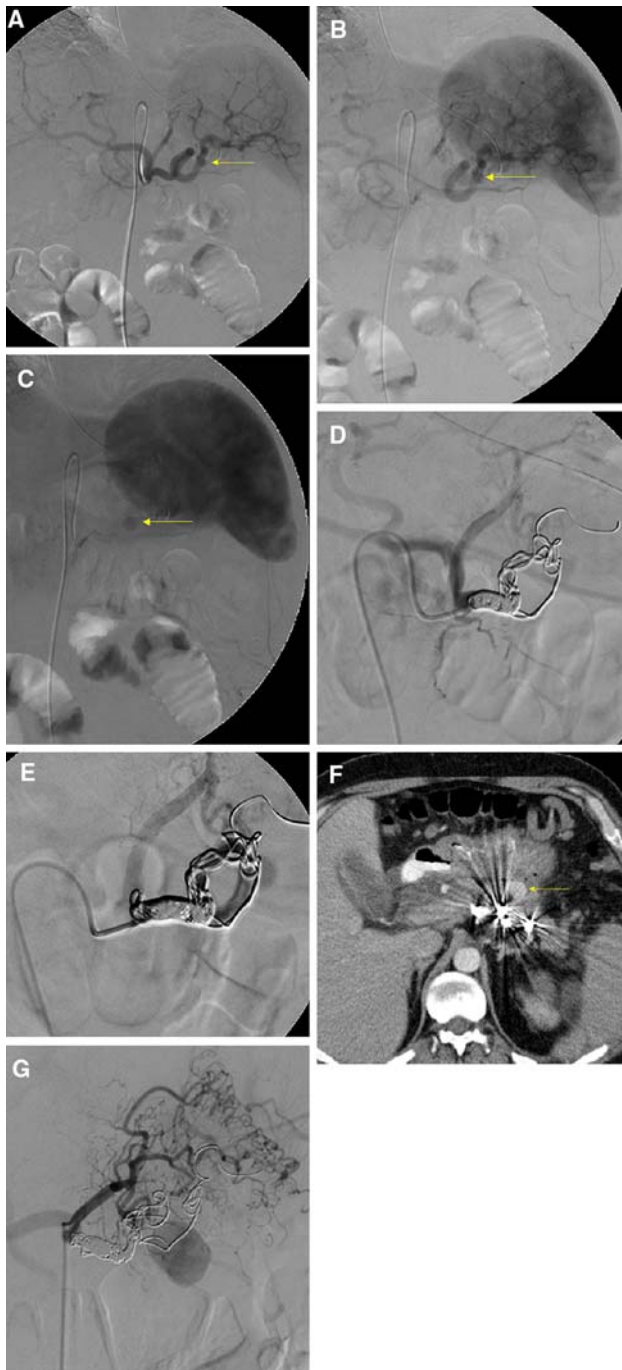


Fig. 15 A 38-year-old male with acute- on- chronic pancreatitis, likely alcohol related, and previous distal pancreatectomy for ruptured pancreatic pseudocyst; admitted with acute upper GI bleed requiring ICU admission. A small aneurysm in the mid splenic artery (arrows; A–E) was coiled, however, the patient continued to be unstable and CT demonstrated a large pseudoaneurysm (arrow; F)—demonstrated at angiography (4 days after initial embolization) to arise from the left gastric artery(G) — that had not been recognized at initial angiography. This was successfully coiled.

angiography have important limitations and potential pitfalls in diagnosis and follow-up. Transarterial embolization is the treatment of choice for arterial hemorrhage.

The technical challenges of access and embolic agent merit careful consideration.

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